AMENDMENTS TO THE SPECIFICATION

IN THE SPECIFICATION

On page 39, line 18, please replace the original paragraph with the following amended paragraph:

-- **Figure 4a:** Schematic view of the 6.7 kb *USF1* gene. Exons are depicted as thick boxes, UTRs as thinner boxes and introns as lines. Genotyped *USF1* SNPs are marked above the gene with associating SNPs indicated with asterixes. A segment of intron 7 is amplified to show the location of the sequence (black bar), used to generate the 20-mer probe used in the EMSA. Nearby SNPs are indicated with larger font and arrows. Sequence shown is SEQ ID NO: 2 --

On page 39, line 24, please replace the original paragraph with the following amended paragraph:

-- **Figure 4b:** Cross-species conservation and EMSA probes. Two probes were constructed that both were capable of producing a shift in the EMSA; One of length 34 bp (residues 1-18 and 50-53 of SEQ ID NO: 3) and the other 20 bp (residues 1-4 and 24-53 of SEQ ID NO: 3). The 34-mer probe contained all three SNPs from this intron 7 region, whereas the 20-mer probe only contained the critical usf1s2 SNP. Below is shown the cross-species sequence conservation and the consensus

sequence. Y stands for pyrimidine and R for purine. Notably the nucleotide at usf1s2 itself is fully conserved, the risk allele representing the ancestral allele. --

On page 106, please replace the first original paragraph with the following amended paragraph:

-- Two affected FCHL family members exhibiting the susceptibility haplotype and two affected FCHL family members without the haplotype were selected for assessment of *USF1* expression in adipose tissue utilizing the SYBR-Green assay (Applied Biosystems). Two step RT-PCR was done using TaqMan Gold RT-PCR kit according to manufacturers' recommendations. A total of 1μg of RNA was converted to cDNA in a 100 μl reaction of which 1 μl was used in the quantitative PCR reaction. The ratio of USF1 to two housekeeping genes GAPDH and HPBGD was used to normalize the data. The specificity of the reaction was evaluated using a dissociation curve in addition to a no-template control. The following PCR primers were used in separate 10 μl SYBR-Green reactions: For USF1; forward: 5´-ATGACGTGCTTCGACAACAG-3´ (SEQ ID NO: 10), reverse: 5´-GGGCTATCTGCAGTTCTTGG-3´(SEQ ID NO: 11). For GAPDH; forward: 5´-CGGAGTCAACGGATTTGGTCGTAT3´(SEQ ID NO: 12), reverse: 5´-AGCCTTCTCCATGGTGGTGAAGAC-3´(SEQ ID NO: 13). For HPBGD; forward: 5´-AACCCTCATGATGCTGTTGTC-3´(SEQ ID NO: 14), reverse: 5´-TAGGATGATGGCACTGAACTC3´(SEQ ID NO: 15). The reactions were run in triplicate using the ABI Prism 7900 HT Sequence Detection System in accordance with the

manufacturers' recommendations and the data were analyzed using Sequence Detector version 2.0 software. --

On page 111, please replace the second original paragraph with the following amended paragraph:

-- We first determined whether the region of usf1s2 represents a binding site for DNA binding proteins. We constructed two 34-mer probes (**Fig 4b**) (residues 1-18 and 50-53 of SEQ ID NO: 3 and residues 1-4 and 24-53 of SEQ ID NO: 3) containing SNPs usf1s2-4 and allowed them to vary for the two alleles of usf1s2. After incubation with nuclear extract proteins of HeLa cells, both critical sequence variants produced an electrophoretic mobility shift (EMS) on a polyacrylamide gel. To further restrict the potentially functional sequence motif, we performed the EMS analyses using a shorter, 20-mer probe pair that shared with the 34-mer probe the critical most conserved nucleotide sequence. This probe produced a mobility shift, comparable to the 34 bp shift, whereas a similar 20 bp probe representing the sequence containing the other strongly associated SNP usf1s1, located in the 3'UTR of *USF1* did not produce a shift (**Figure 5a**). The binding of the probes to nuclear proteins could be competed using unlabeled specific probe, but not with a non-specific probe (**Figure 5b**). --